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# Passive Epicardial Containment Prevents Ventricular Remodeling in Heart Failure

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*Background.* We examined the effects of passive containment of the cardiac ventricles with a surgically placed epicardial prosthetic wrap on indexes of left ventricular (LV) remodeling in dogs with heart failure.

*Methods.* Heart failure (LV ejection fraction 30% to 40%) was produced in 12 dogs by intracoronary microembolization. Six dogs underwent mid-sternotomy and pericardiotomy with placement of a preformed-knitted polyester device (*Acorn* Cardiac Support Device [CSD], Acorn Cardiovascular, Inc, St. Paul, MN) snugly around the ventricles and anchored to the atrioventricular groove. Six dogs did not undergo surgery and served as controls. Dogs were followed for 3 months prior to sacrifice.

*Results.* In controls, LV end-diastolic volume increased after 3 months (67 ± 12 versus 83 ± 8 ml; $p = 0.04$), while in CSD-treated dogs, it decreased (68 ± 10 versus 61 ± 10 ml; $p = 0.002$). CSD-containment of LV size was associated with increased LV systolic fractional area of shortening, while in controls, fractional area of shortening decreased. CSD-treated dogs also showed amelioration of myocyte hypertrophy and attenuation of interstitial fibrosis compared to controls.

*Conclusions.* In dogs with heart failure, passive epicardial containment of the ventricles with the *Acorn* CSD ameliorates LV remodeling and improves LV systolic function.

(Ann Thorac Surg 2000;70:1275–80)


Heart failure (HF) is a progressive disorder whereby the hemodynamic status of the affected patient worsens over time despite the absence of clinically apparent adverse intercurrent events [1]. This deterioration is accompanied by progressive left ventricular (LV) chamber remodeling [2, 3], a process characterized by loss of functional cardiac units, myocyte hypertrophy and interstitial fibrosis at the cellular level, and by changes in LV size and shape at the global level [4]. Increased LV size and chamber sphericity are determinants of functional mitral regurgitation (MR); an undesirable sequelae of HF [5]. Among clinical indicators of progressive LV remodeling, LV dilation, and increased LV sphericity are sensitive predictors of poor long-term outcome and harbingers of death [6, 7]. Treatment with angiotensin converting enzyme inhibitors and β-blockers improve survival in HF [3, 8, 9], in part, by attenuating LV remodeling. In recent years, several surgical approaches have been implemented with the objective of improving cardiac function and ameliorating LV remodeling in patients with HF [10–12]. These include surgical reduction of LV size advocated by Batista and associates [10], dynamic cardiomyoplasty [11], and mitral valve reconstruction to reduce functional MR [12]. The long-term benefits from these procedures, however, remain uncertain. In this study, we examined whether passive epicardial containment of the cardiac ventricles using a preformed-knitted polyester device (*Acorn* Cardiac Support Device [CSD], Acorn Cardiovascular, Inc, St. Paul, MN) can prevent progressive LV remodeling in dogs with moderate HF.

## Material and Methods

### *Animal Model*

The canine model of chronic HF used in the present study was previously described in detail [13]. In this preparation, chronic LV dysfunction is produced by multiple sequential intracoronary embolization with polystyrene Latex microspheres (70 to 102 μm in diameter) which results in loss of viable myocardium. The model manifests many of the sequelae of HF seen in humans, including marked depression of LV systolic and diastolic function, reduced cardiac output, and increased LV filling pressures [13]. In the present study, 12 healthy mongrel dogs, weighing between 21 and 31 kg, under-

Clif Alferness is an employee of Acorn Cardiovascular, Inc, and Hani N. Sabbah, PhD, has a research grant from Acorn Cardiovascular, Inc.

This article has been selected for the open discussion forum on the STS Web site:

http://www.sts.org/section/atsdiscussion/

Accepted for publication April 17, 2000.

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0003-4975/00/$20.00

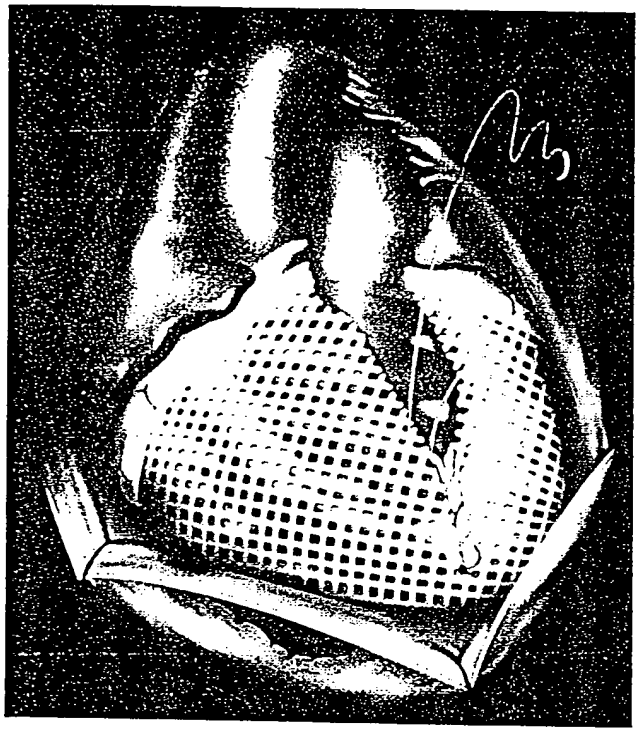

*Fig 1. Artist's depiction of surgical placement of the* Acorn *Cardiac Support Device.*

went coronary microembolizations to produce HF. On average, 6.4 microembolizations were performed in each dog 1 to 3 weeks apart and were discontinued when LV ejection fraction was between 30% and 40%. In this model, this target ejection fraction is associated with a 20% to 30% increase in LV end-systolic and end-diastolic volumes from normal values [3, 13, 14]. Microembolizations were performed during cardiac catheterization under general anesthesia and sterile conditions. The anesthesia regimen consisted of a combination of intravenous injection of oxymorphone (0.22 mg/kg), diazepam (0.17 mg/kg), and sodium pentobarbital (150 to 250 mg to effect) and was previously shown to have no effect on global LV function [3].

### *Study Protocol*

Two weeks after the last coronary microembolization, all dogs underwent a cardiac catheterization (pretreatment). The CSD was surgically implanted in 6 dogs and the remaining 6 dogs did not undergo surgery and served as concurrent controls. In dogs undergoing a CSD implant, anesthesia was induced with intravenous diazepam (0.2 mg/kg) followed by oxymorphone (0.1 mg/kg) and maintained with 0.5% to 1% isoflurane. A median sternotomy was performed, the pericardium opened, and the CSD placed around the ventricles and anchored with approximately eight stay sutures, 2 cm apart, at the atrioventricular groove (Fig 1). The CSD was tailored anteriorly to fit the ventricles snugly. The chest cavity was drained bilaterally with 16F chest tubes and the sternum closed with wires. Prophylactic antibiotics (cefazolin 22 mg/kg) were administered intravenously preoperatively and 4 hours postoperatively. The postoperative course was uneventful in all 6 dogs. All dogs were followed for 3 months during which time no cardioactive drugs were used. At the end of the follow-up period, a cardiac catheterization was performed (posttreatment), the chest was then opened and the heart removed for histologic examination. The study was approved by the Henry Ford Hospital Care of Experimental Animals Committee and conformed to the National Institutes of Health "Guide and Care for Use of Laboratory Animals."

### *Hemodynamic and Angiographic Measurements*

In all dogs, hemodynamic and angiographic measurements were obtained during cardiac catheterizations. LV pressure was measured with catheter-tipped micromanometers (Millar Instruments, Houston, TX). Right ventricular (RV) pressure was measured using a fluid-filled catheter in conjunction with a Perceptor disposable pressure transducer (Schneider/Namic, Glenns Falls, NY). Left ventriculograms were obtained with the dog placed on its right side and recorded on 35 mm cine film at 30 frames/sec during the injection of 20 ml of contrast material (Reno-M-60, Squibb, Princeton, NJ). Correction for image magnification was made using a radiopaque calibrated scale placed at the level of the LV. Left ventricular end-diastolic volume was calculated from angiographic silhouettes using the area-length method. The presence of MR was evaluated qualitatively by noting the degree of opacification of the left atrium during ventriculography. The severity of MR was graded on a scale of 0 to 4+ as previously described [15]. The shape of the LV was quantified from angiographic silhouettes based on the ratio of the major-to-minor axis calculated at end-systole termed "end-systolic sphericity index" [5]. As this ratio decreases (approaches unity), the shape of the LV approaches that of a sphere.

### *Echocardiographic and Doppler Measurements*

Echocardiograms were performed using a model 77030A ultrasound system (Hewlett-Packard, Sonos 1000; Andover, MA) with a 3.5-MHz transducer. Measurements were made with the dog placed in the right lateral decubitus position. In CSD dogs, a parasternal long-axis view was used to measure RV and LV end-diastolic dimensions using M-mode. Measurements were made prior to surgical implantation of the CSD, and 1 week after implantation, to ensure that little or no acute reduction of ventricular dimensions took place during implantation. An LV short axis view at the mid-papillary muscle level was recorded at pre- and posttreatment and used to calculate LV fractional area of shortening (FAS), a measure of LV systolic function. FAS was defined as the difference between the end-diastolic and end-systolic areas divided by the end-diastolic area times 100. To ascertain the presence or absence of constrictive or restrictive physiology in CSD-treated dogs, mitral inflow velocity was measured with pulsed wave Doppler. The following characteristics of mitral flow velocity were measured: (1) peak mitral flow velocity in early diastole (PE); (2) peak mitral flow velocity during atrial contrac-

tion (PA); (3) the ratio PE/PA, and the deceleration time of mitral inflow velocity during rapid early filling (DT).

### *Histologic and Morphometric Assessments*

Once removed, the heart was placed in ice-cold cardioplegia solution. From each heart, LV transverse slices, approximately 3-mm thick, were obtained from the basal, middle, and apical thirds of the LV. The LV free wall from each transverse slice was divided into five transmural blocks, mounted on cork using Tissue-Tek embedding medium (Miles Inc, Mishawaka, IN), rapidly frozen in isopentane precooled in liquid nitrogen. Cryostat sections, approximately 8-$\mu$m thick, were prepared from each block and stained with fluorescein-labeled peanut agglutinin to delineate the myocyte border and the interstitial space including capillaries as previously described [16]. Sections were double-stained with rhodamine-labeled *Griffonia simplicifolia* lectin I (GSL I) to identify capillaries. Ten radially oriented microscopic fields, magnification ×100 (objective 40 and ocular 2.5), were selected at random from each section and photographed using 35-mm color film. Images were projected with a photo magnifier and the cross-sectional area of each myocyte, a measure of myocyte hypertrophy, was calculated using computer-based planimetry. The total surface area occupied by interstitial space and the total surface area occupied by capillaries were measured from each randomly selected field using computer-based video densitometry (JAVA, Jandel Scientific, Corte Madera, CA). The volume fraction of interstitial collagen (interstitial fibrosis) was calculated as the percent total surface area occupied by interstitial space minus the percent total area occupied by capillaries [16]. Capillary density was measured using the index capillary per fiber ratio, and the oxygen diffusion distance was calculated as half the distance between two adjoining capillaries. For comparison, measurements of myocyte cross-sectional area, volume fraction of interstitial fibrosis, and capillary density and oxygen diffusion distance were made employing identical techniques in LV tissue sections obtained from 7 normal dogs. For all of the above measurements, microscopic fields containing scar tissue (infarcts) were excluded. A second set of transverse LV tissue slices was obtained, fixed in formalin, and photographed to grossly visualize the epicardial surface. Each slice was then cut into eight transmural blocks and embedded in paraffin-blocks. Sections were stained with Masson trichrome to delineate fibrous tissue. An average volume fraction of replacement fibrosis, a measure of tissue loss, was calculated for each dog using data obtained from all sections. In CSD-treated dogs, trichrome-stained sections were also used to qualitatively evaluate the extent of epicardial fibrosis resulting from the CSD implantation, as well as to evaluate the interface between the CSD and the epicardial surface of the heart.

### *Data Analysis*

INTRAGROUP COMPARISONS. Comparisons of hemodynamic, angiographic and echocardiographic variables within each of the 2 study groups were made between pretreatment and posttreatment measures. For these comparisons, a Student's paired $t$ test was used, and a probability value less than or equal to 0.05 was considered significant.

INTERGROUP COMPARISONS. To determine whether significant differences in histomorphometric measures were present between the 2 study groups, a $t$ statistic for two means was used with a probability less than or equal to 0.05 considered significant. $p$ Values are reported along with 95% confidence intervals for difference. Data are reported as mean ± standard deviation.

## Results

Autopsy, performed 3 months after implantation, showed a thin, translucent encapsulation of the CSD with connective tissue (Fig 2a). There was no evidence of active inflammation on histologic examination using hematoxylin and eosin staining. Trichrome staining showed encapsulation of the CSD fibers by collagen (Fig 2b, c). The average thickness of the fibrotic layer, including the CSD, was 0.59 ± 0.15. There was a clear demarcation between the CSD and the epicardial surface of the heart with no evidence of invasion of the myocardium by connective tissue (Fig 2c).

Surgical placement of the CSD did not have an acute effect on ventricular dimensions. LV end-diastolic dimension was the same before and 1 week after CSD implantation (4.2 ± 0.5 versus 4.2 ± 0.4 cm). Similarly, RV end-diastolic dimension was the same before and 1 week after CSD implantation (2.3 ± 0.2 versus 2.2 ± 0.2 cm). There was no equalization of LV end-diastolic pressure (13 ± 4 mm Hg) with RV end-diastolic pressure (5 ± 3 mm Hg) at 3 months after CSD implantation. Doppler findings in CSD-treated dogs and in controls are shown in Table 1. The ratio PE/PA increased significantly in controls after 3 months of follow-up but was unchanged in CSD-treated dogs. In CSD-treated dogs, the ratio PE/PA was also unchanged between inspiration and expiration. DT decreased in controls but was unchanged in CSD-treated dogs. Lack of equalization of RV and LV pressures, lack of an increase in the ratio PE/PA or a decrease in DT, and lack of changes in PE/PA during inspiration and expiration suggest the absence of constrictive/restrictive physiology in CSD-treated dogs.

The hemodynamic, angiographic, and echocardiographic measures obtained before and after 3 months of follow-up in both study groups are shown in Table 1. There was no difference in heart rate before and after 3 months of follow-up in either group. LV end-diastolic pressure tended to decrease in CSD-treated dogs and tended to increase in controls, but neither achieved statistical significance. Similar trends were noted with respect to RV end-diastolic pressure. LV end-diastolic volume increased significantly in controls but decreased in CSD-treated dogs. Containment of LV size with the CSD was associated with an increase in LV FAS. In contrast, in control dogs, LV FAS decreased (Table 1).

At baseline, prior to any microembolizations, none of

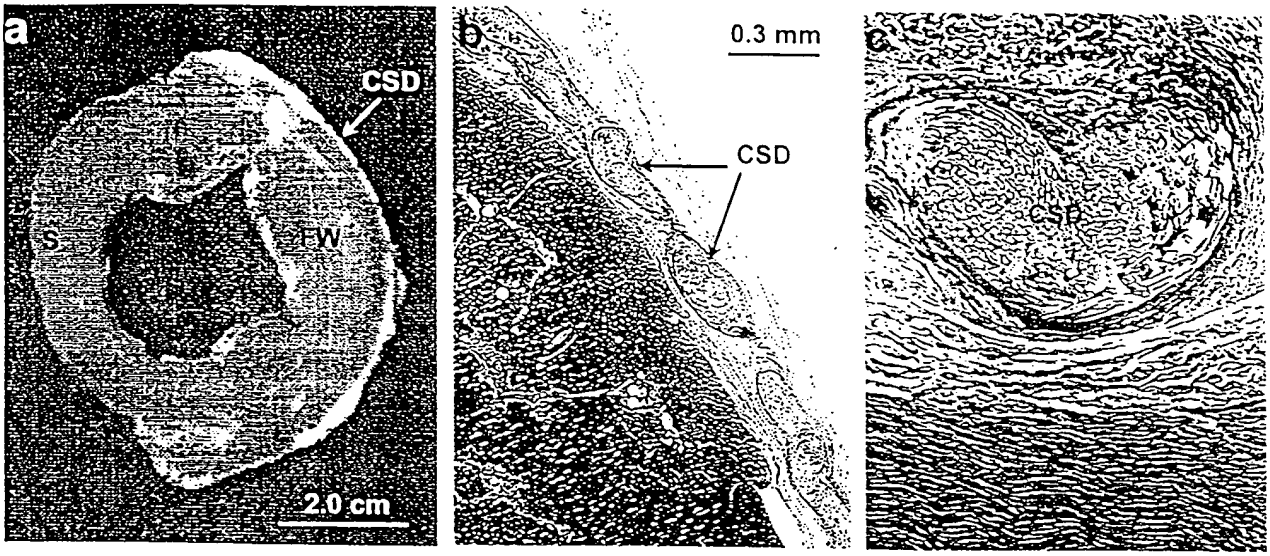


*Fig 2. (a) Transverse slice of left ventricle of a dog treated with the* Acorn *Cardiac Support Device (CSD) for 3 months. The CSD encapsulated by fibrous tissue appears as a white lining along the epicardial surface. (S = interventricular septum; FW = left ventricular free wall.) (b) Trichrome stained section from the left ventricular free wall of same dog as in panel (a) showing a segment of the CSD fibers encapsulated in collagen (blue) and overlying the myocardium (red). (c) Trichrome stained section showing a magnified view of a single CSD fiber encapsulated in collagen (blue). Note the clear demarcation between the CSD and myocardium (red).*

the 12 study dogs manifested MR measured using Doppler. Ventriculograms showed that 4 of 6 control dogs developed mild to moderate (1+ to 2+) MR (average severity, 0.83 ± 0.31) before initiating therapy that persisted or worsened (1 of 4 dogs) after 3 months of follow-up (average severity, 1.00 ± 0.37) ($p = 0.73$). Four of 6 CSD-treated dogs also developed mild to moderate (1+ to 2+) MR (average severity, 1.00 ± 0.37) prior to CSD implantation. In contrast to controls, the MR was completely abolished in CSD-treated dogs after 3 months of follow-up ($p = 0.021$). The elimination of functional MR in CSD-treated dogs was associated with an increase in the LV end-systolic sphericity index. In contrast, the sphericity index was unchanged in controls (Table 1).

In addition to improving LV systolic function and limiting functional MR, prevention of progressive LV enlargement in CSD-treated dogs had a beneficial effect on cellular components of LV remodeling (Table 2). In CSD-treated dogs, myocyte cross-sectional area was smaller than in controls as was the volume fraction of interstitial fibrosis. Treatment with the CSD was also associated with a higher capillary density and a lower oxygen diffusion distance (Table 2). Overall LV replacement fibrosis tended to be lower in CSD-treated dogs

*Table 1. Angiographic, Echocardiographic, and Doppler Measures Obtained Before (Pre) and After 3 Months of Follow-up (Post) in Both Study Groups*

| | Control | | | | *Acorn* CSD | | | |
|---|---|---|---|---|---|---|---|---|
| Variable | Pre | Post | *p* Value | 95% CI | Pre | Post | *p* Value | 95% CI |
| HR (beats/min) | 79 ± 17 | 72 ± 6 | 0.22 | −3.19 to 19.86 | 85 ± 8 | 84 ± 11 | 0.81 | −10.77 to 13.10 |
| LV EDP (mm Hg) | 14 ± 5 | 17 ± 66 | 0.19 | −7.68 to 2.02 | 15 ± 3 | 13 ± 4 | 0.22 | −1.51 to 5.18 |
| RV EDP (mm Hg) | 6 ± 1 | 7 ± 2 | 0.22 | −3.31 to 0.98 | 7 ± 1 | 5 ± 3 | 0.14 | −0.62 to 3.29 |
| LV EDV (ml) | 67 ± 12 | 83 ± 20 | 0.036 | −28.86 to −1.48 | 68 ± 10 | 61 ± 10 | 0.002 | 3.88 to 9.46 |
| LV FAS (%) | 27 ± 3 | 23 ± 4 | 0.012 | 1.35 to 6.65 | 29 ± 5 | 32 ± 4 | 0.010 | −6.62 to −1.40 |
| LV ES sphericity index | 1.40 ± 0.14 | 1.38 ± 0.15 | 0.43 | −0.03 to 0.07 | 1.48 ± 0.11 | 1.64 ± 0.20 | 0.038 | −0.29 to −0.01 |
| PE (cm/sec) | 62 ± 14 | 61 ± 11 | 0.80 | −10.16 to 12.50 | 65 ± 15 | 48 ± 4 | 0.040 | 0.66 to 34.34 |
| PA (cm/sec) | 27 ± 7 | 21 ± 6 | 0.003 | 2.95 to 8.05 | 32 ± 10 | 23 ± 4 | 0.08 | −1.74 9 to 19.86 |
| PE/PA | 2.4 ± 0.4 | 3.0 ± 0.6 | 0.016 | −1.06 to −0.17 | 2.2 ± 0.8 | 2.1 ± 0.5 | 0.70 | −3.19 to 19.86 |
| DT (msec) | 95 ± 10 | 79 ± 9 | 0.047 | 0.37 to 32.96 | 91 ± 11 | 94 ± 8 | 0.28 | −3.19 to 19.86 |

CI = confidence interval of the difference; CSD = cardiac support device; DT = deceleration time of mitral inflow velocity during rapid early filling; EDP = end-diastolic pressure; EDV = end-diastolic volume; ES = end-systolic; FAS = fractional area of shortening; HR = heart rate; LV = left ventricular; PA = peak mitral flow velocity during atrial contraction; PE = peak mitral flow velocity in early diastole; PE/PA = the ratio PE/PA; RV = right ventricular.

*Table 2. Morphometric Measures of Cellular Remodeling Made 3 Months After Follow-up in Both Study Groups*[a]

| Variable | Normal | Control | *Acorn* CSD | *p* Value | 95% CI |
|---|---|---|---|---|---|
| MCSA ($\mu m^2$) | 616 ± 44 | 955 ± 87 | 791 ± 125 | 0.025 | 24.91 to 303.09 |
| VFIF (%) | 3.5 ± 0.7 | 12.2 ± 0.9 | 10.2 ± 1.5 | 0.018 | 0.42 to 3.58 |
| Capillary density | 1.00 ± 0.05 | 0.95 ± 0.01 | 1.14 ± 0.13 | 0.003 | −0.30 to −0.08 |
| ODD ($\mu m$) | 11.8 ± 0.3 | 13.5 ± 0.6 | 11.8 ± 0.6 | 0.0001 | 0.97 to 2.43 |
| VFRF (%) | ... | 19 ± 4 | 17 ± 7 | 0.73 | −6.03 to 10.03 |

[a] *p* Values are based on comparisons of Control and *Acorn* CSD groups, as is CI.

CI = confidence interval of the difference; CSD = cardiac support device; MCSA = myocyte cross-sectional area; ODD = oxygen diffusion distance; VFIF = volume fraction of interstitial fibrosis; VFRF = volume fraction of replacement fibrosis.

compared to controls, but the difference did not reach statistical significance (Table 2).

## Comment

The observations made in this study indicate that passive epicardial containment of the cardiac ventricles with the *Acorn* CSD prevents progressive LV dilation in dogs with moderate HF. This benefit occurred in the absence of any hemodynamic or Doppler-echocardiographic evidence of restrictive/constrictive physiology. An unexpected, yet consistent finding, was the observation of improved LV systolic function in CSD-treated dogs evidenced by an increase in LV FAS. Prevention of progressive LV dilation was also associated with attenuation of myocyte hypertrophy and interstitial fibrosis. Accumulation of collagen in the interstitial space can have a direct impact on myocyte function and viability. We previously showed that in the failing heart, LV regions that manifest severe interstitial fibrosis may be subjected to chronic hypoxia [17]. The increase in capillary density and the reduction in oxygen diffusion distance seen in the present study in CSD-treated dogs argue in favor of improved function and viability of constituent myocytes of the failing heart. While use of the CSD appears to be beneficial, a possible downside is the difficulty in performing coronary artery bypass surgery in the future, given the limited by access to epicardial arteries due to fibrosis that encapsulates the CSD and its adherence to the epicardial surface of the heart. Exploration of approaches that allow the performance of coronary artery bypass surgery, once the CSD has been implanted and fibrosis established, is currently under investigation.

Another potential benefit of the CSD is the elimination of functional MR, albeit at mild-to-moderate severity. When present, MR can play a role in reducing LV stroke output and can contribute to worsening of the HF state. The clinical importance assigned to this functional abnormality is exemplified by the recent interest in surgical mitral valve reconstruction in patients with HF and severe functional MR [12]. Functional MR in the setting of HF is frequently attributed to LV enlargement, mitral annular dilation and, more importantly, to changes in LV shape manifested by increased chamber sphericity [5]. Use of the CSD decreased chamber sphericity with a subsequent elimination of functional MR.

It is fair to speculate that dynamic cardiomyoplasty gave rise to the concept of passive ventricular containment as a potential therapeutic modality for preventing progressive LV dilation in HF. Studies in patients by Kass and colleagues [11] suggested that the benefits derived from cardiomyoplasty, in terms of reverse LV remodeling, may be primarily due to passive girdling of the heart rather than to an active squeezing assist effect. Studies in patients with HF [18], and in sheep with myocardial infarction [19], that underwent dynamic cardiomyoplasty also showed that the efficacy of the procedure may be due to a passive "girdling effect" which limits the progression of ventricular enlargement; augmentation of systolic function through active contraction of the latissimus dorsi may be only a secondary phenomenon. In dogs with HF secondary to rapid ventricular pacing, wrapping the ventricles snugly with a Marlex mesh (C.R. Bard, Inc, Murray Hill, NJ) was effective in preventing cardiac enlargement but not as effective as adynamic cardiomyoplasty, possibly due to adaptive capabilities of the skeletal muscle [20]. In dogs with doxorubicin-induced HF, cardiac binding with polytetrafluoroethylene prevented ventricular dilation but without eliciting improvement of LV systolic function [21]. Both studies are limited by having performed cardiac binding prior to inducing HF and, therefore, are difficult to interpret in light of the findings of the present study.

There are some limitations to the study that warrant consideration. The canine model of HF used in this study manifests diffuse infarcts which lead to global LV dysfunction and HF. As such, it differs from clinical situations where HF results from a large regional transmural myocardial infarction. Additional studies are needed to determine the efficacy of the CSD in these circumstances. Another limitation is the lack of a sham-operated group in which the pericardium was opened and left open. The study was conducted for only 3 months and in dogs with moderate HF (LV ejection fraction 30% to 40%). Additional studies are needed to assess the efficacy of the CSD over longer periods of time and in animals with advanced HF. In the present study, only monotherapy with the CSD was examined. Whether or not the CSD can elicit improvements, above and beyond those seen with prototypical drugs used for the treatment of HF, remains uncertain and requires further evaluation.

In conclusion, passive epicardial containment of the cardiac ventricles with the *Acorn* CSD prevents progressive LV remodeling in dogs with moderate HF. Prevent-

ing LV dilation appears to attenuate the adverse effects of LV remodeling and prevent functional MR. Additional studies are needed to further elucidate the mechanisms through which ventricular constraint with the *Acorn* CSD elicits its benefits in HF. Nevertheless, the findings of this study warrant consideration of this surgical approach as adjunct in the management of chronic HF.

This study was supported, in part, by a grant from Acorn Cardiovascular, Inc, and by a grant from the National Heart, Lung and Blood Institute, HL49090-06.

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# Differences between wrapping and assist devices relevant to remodeling

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**Differences between wrapping and assist devices relevant to remodeling**
By Ray Chu-Jeng Chiu on Wed Nov 15, 2000 04:00 EST

Dr. Chaudhry and associates reported very interesting and highly favorable data regarding the efficacy of passive constraints of the cardiac ventricles using an epicardial prosthetic wrap, in a model of heart failure produced by intra-coronary micro-embolization. Not only were there signs of ventricular reverse remodeling, but also improvement in systolic function. Importantly, they reported no evidence of ventricular constriction. The major advantage of this approach as compared to cardiomyoplasty is that the prosthetic ventricular wrapping is a less invasive operation. One which would be relevant in its clinical application is the ability for auto-regulation by the "contained heart" to acute changes in the filling pressure. With limited stretchability of the prosthetic wrap, even though it does not show constrictive physiology under a steady state, a sudden increase in filling pressure either as a physiological response or as the result of therapeutic intervention, may be poorly tolerated by the rapid rise in left ventricular end-diastolic pressure, possibly leading to pulmonary edema. In future studies, observing the response of contained ventricles to rapid volume expansion would be worthwhile.

One exciting but somewhat perplexing finding is that a contained heart in fact became smaller in dimension, while improvement of myocardial damage at the cellular level is noted. Although the authors pointed out the similarity between these observations with those reported in patients following prolonged mechanical cardiac assist, in fact, such a prosthetic wrap does not by itself reduce preload to decreased ventricular size, as can be achieved with mechanical cardiac assist devices. The data reported here would imply that even though the myocardial tension, a major signal for the remodeling process, is not reduced actively by the prosthetic wrapping, the cardiac muscle can spontaneously heal if there is no progressive dilatation of the ventricles. Would the ability for the cells to heal be related to the etiology of the myocardial damage and heart failure? For example, would the idiopathic or viral cardiomyopathic myocytes heal like those in an ischemic cardiomyopathy? As the authors concluded, further studies are warranted.

Ray C.J. Chiu,MD, PhD

McGill University, Montreal

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# The case for a multimodal surgical approach to heart failure

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**The case for a multimodal surgical approach to heart failure**
By Steven F Bolling on Wed Nov 15, 2000 07:00 EST

Thank you very much for allowing me to comment on the manuscript entitled, Passive Epicardial Containment Prevents Ventricular Remodeling in Heart Failure, by Dr. Chaudhry et al. from Dr. Sabbah's laboratory at Henry Ford. In this well-done study, the authors looked at the effects of a passive constraint device on the cardiac ventricles in a very nice heart failure model. They showed that in dogs with heart failure, passive epicardial containment with the Acorn constraint device improves left ventricular reverse "remodeling" and improves left ventricular systolic function.

In comment, one must say that this cardiac constraint device will be one of a series of surgical mechanical devices to alter left ventricular geometry in heart failure. Many types of surgeries, including cardiomyoplasty, the Batista operation, and undersized mitral valve reconstruction have been attempted to alter left ventricular geometry. This Acorn constraint may be a useful adjunct in a continuation of the cardiomyoplasty idea, in terms of diastolic containment.

The results from this study, as well as other preliminary studies with this device, have been quite encouraging in terms of reverse remodeling and improvements in left ventricular systolic function. The cardiac surgeon's natural hesitation to use a device such as this will be based on concerns regarding long-term epicardial and pericardial fibrosis with constriction and possible negative impacts on diastolic function. These patients can ill-afford any reduction in their already poor diastolic function, and long-term studies may be needed to clarify this point. However, initial results are encouraging. This device may also be thought of as complementary to other types of higher risk conventional surgeries on heart failure patients, including high-risk coronary artery bypass grafting and high-risk mitral valve reconstruction. In this era, when limitations upon transplantation (financial and the donor availability) are increasing, it is encouraging to see innovative types of surgical therapy being developed for these patients, who would otherwise be relegated strictly to "more medicines". While certainly medical therapy for congestive heart failure has moved forward rapidly over the last five years, a complementary approach of both surgery and medicine individually suited for the patient will be the best answer. In summary, this type of passive constraint device to effect favorable reverse remodeling or to inhibit worsening of the LV bad remodeling may be useful for the future. One must remain excited about the possibility of this and other types of devices.

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